therefore be estimated to be well in excess of 400. This compares with the present number of 349 consultants in England and Wales, and indicates the probability of expansion rather than contraction in the specialty.

Of the 349 consultants in diseases of the chest, 242 (nearly 70%) are stated to be over the age of 50, and only two below the age of 40. This peculiar age-distribution is consequent upon the contraction in the specialty which followed the decline of tuberculosis. But other respiratory diseases continue unabated, or even increasing in relative importance; and, unless recruitment to training in the specialty is expanded, within 15 years there will be a grave deficiency of consultant physicians trained in chest diseases. It is therefore to be hoped that the Ministry's gloomy assessment of prospects will not discourage recruitment to training in what appears likely to be an expanding specialty.—We are, etc.,

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Radiation Hazards of High Altitude Flight

SIR,—Few of your many medical readers will be called upon to advise on "longer space voyages" and so to recall your very topical leading article (21 December, p. 719). The omission of mention of the high concentration of electrons in the Van Allen[1] belts is therefore of little consequence. Your readers may, however, soon be asked about radiation in high altitude flight in Concorde, etc. Other experts are not nearly so sanguine as yours on the practicability of prediction of solar flares. The Space Radiation Study Panel[2] and a Task Group of Committee I of the International Commission on Radiological Protection[3] suggested that the vast majority of visible solar disturbances do not result in significant radiation near the earth; those that do, and notably those that result in potentially dangerous giant flares, are not distinguishable from the others. The likely warning period is not always the 24 hours you give but may be as little as 10 minutes.—I am, etc.,

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Factors in Host–Virus Relationship

SIR,—The interesting paper "For Debate," by Dr. H. E. Webb (14 December, p. 684) raises many controversial points. In relation to his suggestion that dual virus infections may produce unusual clinical manifestations, I wish to continue the debate by pointing to some evidence which might incriminate the following dual virus infections in certain diseases.

Measles and herpes simplex are both common infections of early childhood. Both viruses produce stomatitis, and both are potentially neurotropic. If herpes stomatitis were superimposed on a measles stomatitis it would probably not be detected clinically, and a dual infection would not be suspected. It seems possible that primary infection with herpes simplex (a D.N.A.-virus) occurring during the acute stage of a primary infection with measles (an R.N.A. virus) might so modify the measles infection, perhaps by production of interferon, that in some cells production of infective measles virus might be defective or incomplete. These cells might survive and replicate with accompanying replication of incomplete virus. If such were the case infective virus would not be found, but antibody might be produced as has been shown in incomplete infections of certain mammalian cell lines by influenza virus.[1] That such a dual infection with measles and herpes simplex might result in subacute sclerosing panencephalitis (S.S.P.E.) finds some support from the serological findings reported by Legg[2] in 22 patients and 23 controls; not only were there high antibody levels for measles in the S.S.P.E. patients, but also complement-fixing antibody for herpes simplex (but not for mumps, lymphocytic choriomeningitis, influenza A, influenza B, influenza C, and adenovirus) was found in a significantly greater number of the patients than the controls.

Recent reports suggest that herpes simplex and varicella-zoster viruses are antigenically related.[3,4] These are well-recognized potentially latent viruses, and their cell membranes contain host material.[5] Although the majority of the adult population have been infected with both of these viruses only a small proportion of such individuals have recurrent herpes simplex or develop zoster. It seems possible that subsequent attacks of recurrent herpes simplex or of zoster may be determined by which of these two viruses causes the first infection, and also perhaps by the interval between the two primary infections. If this interval is at least several weeks, and if the first infection in an individual is herpes simplex, that virus will become latent after the acute infection has subsided; it seems possible that latency of subsequent infection with varicella-zoster might be prevented by the boosting of the antigenically related herpes simplex antibody. Thus such an individual may be liable to attacks of recurrent herpes simplex but not to zoster. Similarly, if varicella is the first infection followed thereafter by herpes simplex, the varicella-zoster virus may become latent and thereafter cause zoster, but that individual might be free from recurrent herpes simplex infections. This possible relationship could, of course, be substantiated by showing that attacks of recurrent herpes simplex and zoster generally affect different individuals—an ideal investigation for general practice. If by coincidence there is combined active infection—that is, a dual infection—with two antigenically related viruses such as herpes simplex and varicella-zoster, or measles and an as-yet-unknown related human pathogen, it seems possible that there might be interference with the normal development of specific IgM and IgG antibodies. Such a dysfunction might explain the pathogenesis of conditions such as S.S.P.E., or multiple sclerosis, where there are high antibody levels to various viral agents accompanied by abnormalities in immune globulins.—I am, etc.,

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Inhibition of Lactation with Oestrogens

SIR,—With respect I must disagree with Professor D. Llewellyn-Jones (9 November, p. 387) when he states that "inhibition of lactation using oestrogens is still the treatment of choice."

Three years ago I reported[1] that we were using no oestrogens and were getting perfectly satisfactory results by encouraging an extra fluid intake during the first three days. The rationale of this treatment is that engorgement is detrimental to breast feeding.[2] Waller used small doses of oestrogens to damp down engorgement, and this increased the mother's milk yield. Giving extra fluids may increase engorgement and thus be harmful to lactation.

During the last three years we have kept records and have learned that in 217 patients whose lactation was suppressed with water 55 had no discomfort, 50 slight, and 53 fair to moderate discomfort. Twenty-one had quite severe discomfort. This severe discomfort is adequately treated with analgesic drugs. Aspirin, Panadol (paracetamol), D.F. 118 (dihydrocodeine bitartrate), codeine, and Zactirin (tablets containing ethohaptazine citrate) have all been used according to the whim of the residents. In 109 instances, however, no analgesic was required or given.

It has of course been suggested that the giving of extra fluids cannot influence the result. However, in a group given no extra fluids the results were not so good.

In 106 patients who had been given extra fluids lactation had ceased,

by the	3rd	day	in	94 patients
„ „	4th	„	„	1 patient
„ „	5th	„	„	2 patients
„ „	6th	„	„	2 „
„ „	7th	„	„	1 patient

In three instances lactation was not suppressed.

In a group of 42 patients who had been given no treatment at all,

31	ceased	lactation	in	hospital
2	„	„	at	12 days
1	„	„	„	2 weeks
1	„	„	„	4 „
5	„	„	„	5 „
2	„	„	„	6 „

It does seem that the promotion of engorgement is a help in suppressing lactation.

I agree with Mr. S. J. Steele (30 November, p. 578) that oestrogens need not be used, and, since there is the unpleasant suggestion that thrombosis may be promoted, then I would go further and say that they ought